CASE REPORT

HEPATITIS A INFECTION: AN UNUSUAL CAUSE OF PLEURAL EFFUSION

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Abstract

Jaundice due to hepatitis A virus is an important health problem especially in developing countries. However, this self limiting illness can sometimes present with atypical features. We report a four year male child who presented to us with ascites and pleural effusion secondary to Hepatitis A. The child improved with conservative treatment.

Keywords: hepatitis A, pleural effusion, ascites

Introduction

Hepatitis A is widely prevalent in developing countries, mode of transmission being feco-oral route. Its clinical presentation may vary from anicteric hepatitis to fulminant hepatic failure. It is a benign, self limiting disease but may present with atypical clinical findings. Extrahepatic manifestations are reported in 6.4-8% cases. These are arthralgia, cutaneous vasculitis, cryoglobulinemia, hemophagocytic syndrome, acalculous cholecystitis, pancreatitis, aplastic anemia, Gullain Barre syndrome, transverse myelitis, vasculitis, ascites and pleural effusion. (1,2) Among these pleural effusion and ascites are rare complications of hepatitis A, especially in childhood. These occur during early period of disease and resolve spontaneously with resolution of hepatitis. (3-5) Herein we present a case of acute hepatitis A complicated by pleural effusion and ascites.

Case Report

A four year male child was admitted to our hospital with complaints of generalized swelling all over the body, abdominal distension and difficulty in breathing for 8 days. Further, mother informed that child had been unwell over the last one month. Illness started with low grade fever lasting for 15 days which was associated with poor appetite, yellowish discoloration of eyes and urine. For these complaints patient was admitted at another hospital where investigations showed bilirubin - 3.5 mg/dl (direct- 2.2 mg/dl), ALT-560 IU/l, AST-460 IU/l, alkaline phosphatase-1045 IU/l. Urine was positive for urobilinogen. Chest X ray showed bilateral pleural effusion. Patient was referred to us for further management after five days of hospitalization. On presentation, he had pallor, generalized edema but no jaundice. Abdomen examination revealed ascites with hepatomegaly. On auscultation of chest, breath sounds were decreased on right side of chest. Laboratory studies revealed a normal hemogram. Liver function test showed a total serum bilirubin-0.44 mg/dl (direct bilirubin-0.28 mg/dl), AST-52.6 U/L, ALT-38.9 U/L, alkaline phosphatase -110 U/L. Total serum protein was 7.5 gm/dl, serum albumin was 4.5 gm/dl. Renal function test and serum electrolyte were normal. Coagulation studies were normal. Chest X ray showed bilateral pleural effusion more on right side. Serological analysis for dengue, enteric fever, leptospirosis and rickettsial infections were negative. IgM antibody for hepatitis A was positive. Other viral markers including hepatitis B, C and E were negative by Elisa. Ultrasound (USG) guided thoracocentesis was undertaken and pleural fluid analysis was suggestive of transudative effusion. Pleural fluid for tuberculosis polymerase chain reaction (TB-PCR) and culture were negative. An abdominal USG confirmed the presence of ascites. Patient received supportive treatment in the form of dietary advice, rest and silymarin. Repeat chest X ray after 8 days of admission was normal. Repeat abdominal sonography showed only streak fluid in subhepatic region. Child is on regular follow up and is doing well.

Discussion

Hepatitis A is usually self limited in children and 85% patients recover completely. Mortality rate increases with age. (6) Pleural effusion as an early complication of hepatitis A has been reported previously, the first case being reported in 1971. (7) There are only few cases of hepatitis A with pleural effusion and ascites reported worldwide. (8) The exact etiopathogenesis of pleural effusion is unknown, but it may be attributed to inflammation of liver, immune complexes or secondary to ascites due to transport of fluid from diaphragmatic lymphatics or secondary to hypoalbuminemia. Ascites may occur as a result of venous and lymphatic obstruction or decrease in the osmotic pressure of plasma colloid. (6-8) A transient increase in portal venous or lymphatic pressure due to compression of hepatic sinusoids may explain the occurrence of ascites. (9,10) As liver function improves, ascites and pleural effusion resolve spontaneously as seen in our patient. (11)

Conclusion

Although hepatitis A infection is known to be a benign self limiting illness, sometimes it may present with unusual complications posing diagnostic dilemma. Clinicians must be aware of this entity to avoid unnecessary investigations.

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